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Anti-Ulcer Activity of Lansoprazole in Experimental Ulcer Models

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Abstract: *Lansoprazole is considered a second-generation proton pump inhibitor, used for target-specific acid suppression by inhibiting the parietal cell H+/K+-ATPase . But there is evidence that its clinical impact is not only from acid control , it also involves non-acid dependent cytoprotective mechanisms, kind of like additional safeguard routes. This systematic study tried to map that dual-action profile, by weighing its antisecretory strength against its own antioxidant and tissue remodeling effects across five well-validated rodent models of gastric injury.*

Adult Wistar rats were tested under five separate mucosal stressors: surgical pylorus ligation, absolute ethanol exposure, indomethacin dosing, water-immersion restraint stress , and serosal acetic acid application. In the pylorus ligation setup, 20 mg/kg Lansoprazole gave a significant ($p < 0.01$) protection rate of 84.37% , and it lowered gastric juice volume and total acidity , while raising pH. Then, in that acid-independent ethanol model, it curtailed the linear hemorrhagic necrosis down to smaller pinpoint petechiae, reaching 80.41% protection. It also kept strong mucosal defenses when prostaglandins were depleted (79.08% protection) and during neurogenic stress (81.07% protection). Plus, it sped up chronic ulcer healing overall, with 85.52% recovery.

On the biochemical side, Lansoprazole reduced cell membrane lipid peroxidation , keeping malondialdehyde levels low. It also prevented depletion of endogenous antioxidant reserves, meaning SOD, catalase, and glutathione were held better ($p < 0.01$). Histopathology backed this up, showing that Lansoprazole preserved epithelial integrity, reduced submucosal edema and, importantly, suppressed the pro-inflammatory neutrophil inflow.

So, in conclusion, Lansoprazole seems to coordinate target-directed proton pump blockade with its intrinsic radical-scavenging capacity, and it adds prostaglandin independent systemic defense support, while also pushing faster structural tissue repair. All of this together supports its multi-pronged therapeutic efficacy , and honestly it looks pretty robust across the models used.

Keywords: Lansoprazole; Gastric Mucosal Defense; Proton Pump Inhibitors; Antioxidant Enzymes; Lipid Peroxidation; Cytoprotection; Histopathology

I. INTRODUCTION

Peptic ulcer disease is still a big epidemiological and clinical problem in modern gastroenterology. It affects millions of people worldwide and also drags down healthcare resources, mainly because of complications like upper gastrointestinal bleeding, perforation , and gastric outlet obstruction, you know the usual bad outcomes. On the mechanistic side, keeping the gastric mucosa “intact” depends on this kind of balancing act between bad luminal forces and also defensive mucosal barriers, and if that balance goes off then trouble starts.

The “aggressive” side is made up of endogenous elements like hydrochloric acid and pepsin, plus exogenous threats such as Helicobacter pylori infection, chronic non-steroidal anti-inflammatory drugs (NSAIDs) use, heavy alcohol consumption, and both physiological or even psychological stress. Meanwhile the defensive barrier is kind of layered, not just one thing. There is a pre-epithelial zone (a hydrophobic mucus-bicarbonate gel barrier), then an epithelial layer

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with tight cellular junctions, quick cell turnover, and ongoing surface renewal. After that, there is a post-epithelial component—dense submucosal blood flow, which brings oxygen and nutrients and also helps clear back-diffusing hydrogen ions. When those natural defenses get overrun, acute or chronic mucosal erosion can occur, and then peptic ulcers form.

Now, looking at endogenous aggressive forces , hydrochloric acid secreted by parietal cells is sort of a necessary medium for peptic activity and auto-digestion. Acid production is a highly regulated physiological process. It is controlled by histamine, acetylcholine, and gastrin through parallel intracellular signaling routes. Those routes converge onto the terminal step of hydrogen ion transport: the gastric proton pump, also called hydrogen-potassium adenosine triphosphatase (H+/K+-ATPase).

Since acid is considered a primary driver for ulcer development, reducing gastric acid output has been the main strategy for years in managing acid-related disorders. That includes duodenal ulcers, benign gastric ulcers, gastroesophageal reflux disease (GERD), and hypersecretory conditions like Zollinger-Ellison syndrome, where secretion is abnormally high.

Treatment for peptic ulcer disease has also changed over time: it went from basic antacids to histamine H2-receptor antagonists, and now the current standard is proton pump inhibitors (PPIs). Drugs like omeprazole, lansoprazole, pantoprazole, and rabeprazole are lipophilic weak bases. They're given as inactive prodrugs, and once they get into systemic circulation, they cross cellular membranes selectively and build up in the super acidic secretory canaliculi of active gastric parietal cells, so the local pH falls below 2.0.

That localized drop in pH makes the PPI molecule undergo a fast, acid-catalyzed rearrangement into a reactive electrophilic tetracyclic sulfenamide intermediate. This active intermediate then forms covalent disulfide bonds with available sulfhydryl groups on specific cysteine residues found within the extracellular domain of the catalytic α-subunit of the H+/K+-ATPase pump. This covalent change basically shuts the enzyme down irreversibly, which blocks the final hydrogen ion transport step into the gastric lumen. As a result, it suppresses both basal and stimulated acid secretion.

$$\text{Enzyme-SH} + \text{PPI-Sulfenamide}^{+} \longrightarrow \text{Enzyme-S-S-PPI} + \text{H}^{+}$$

Lansoprazole is like a prominent second-generation substituted benzimidazole derivative in this drug class, kind of, yeah. It shows high absolute oral bioavailability right from the start dose and it also comes with rapid systemic absorption kinetics. Even though its plasma elimination half-life is rather short, about 1.3 to 2.1 hours, the pharmacodynamic clock for acid suppression still lasts roughly 24 to 48 hours. That longer effect really comes from irreversible covalent binding onto the proton pump, so acid output stays suppressed until fresh enzyme molecules are built again by the parietal cells, you know.

In practice, Lansoprazole's quick onset and the sustained anti-secretory strength make it a core piece of therapeutic plans, especially when it is paired with antibiotics for Helicobacter pylori eradication, and also as monotherapy if the goal is preventing , or healing NSAID-related gastric erosions. While the main anti-secretory mechanism is pretty well established, newer preclinical clues hint that the therapeutic worth goes beyond just acid lowering. Some recent work suggests that substituted benzimidazoles have pleiotropic, non-acid-dependent cytoprotective and tissue-stabilizing advantages, and these seem to run independently of parietal cell pump inhibition.

Those extra actions are thought to include direct free-radical scavenging, safeguarding the natural tissue antioxidant network, reducing pro-inflammatory cytokine cascades, and even speeding up structural tissue remodeling. Under conditions of intense chemical or ischemic stress, the gastric mucosa ramps up a surge of reactive oxygen species, like superoxide anions, hydrogen peroxide, and hydroxyl radicals. Once that radical burst hits, it can push cell membrane damage through lipid peroxidation, so membrane integrity gets disrupted and that can escalate into deeper tissue necrosis.

Lansoprazole has also been shown to increase cellular survival pathways, including the Nrf2/HO-1 signaling pathway, which helps keep the native antioxidant enzyme pools such as Superoxide Dismutase (SOD), Catalase (CAT), and Reduced Glutathione (GSH). To fully pin down this "two-sided" profile, the drug really needs assessment across

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multiple preclinical model systems that mimic different pathological routes. A lot of studies screen ulcer candidates using only one injury model , which can miss non-acid-dependent mechanisms or fail to reflect what happens when endogenous defensive systems are already impaired.

For example, if a compound is judged only by surgical pylorus ligation, you capture its anti-secretory ability, but it still can't really tell its direct cytoprotective performance against non-acid-dependent chemical necrosis, like the kind triggered by absolute ethanol exposure. Likewise, just running chemical challenges doesn't expose how the drug behaves during neurogenic stress, or during systemic prostaglandin depletion either.

So, to close those gaps, this systematic investigation looked at the multipronged pharmacological profile of Lansoprazole across five separate validated in vivo rodent models of gastric injury, which were carefully selected.

1. Absolute Ethanol-Induced Model: Here the mucosa gets challenged through direct chemical necrosis, and that stripping away of protective mucus happens along with microvascular stasis, independent of acid concentration. This setup is basically a clean way to test non-acid-dependent cytoprotection.
2. Indomethacin-Induced Model: This imitates NSAID-type injury by non-selectively inhibiting the COX-1 enzyme, depleting cytoprotective prostaglandins, and leaving the tissue more exposed to back-diffusing acid.
3. Water-Immersion Restraint Stress (WIRS) Model: It represents a layered neurogenic process, where physical restriction plus cold exposure leads to sympathetic vasoconstriction (ischemia) while parasympathetic hypersecretion kicks in too.
4. Surgical Pylorus Ligation Model (Shay Method): It forces an unbuffered accumulation of endogenous gastric juices, and that allows tight measurement of fluid volume shifts, pH changes, and total acid output.
5. Serosal Acetic Acid-Induced Model: This creates a localized, full-thickness chronic ulcer crater that resembles human peptic ulcer disease, giving a setup to study longer-term repair, re-epithelialization, and glandular remodeling across an extended timeline.

By combining quantitative datasets from those five distinct challenge streams, including macroscopic lesion scoring, micro-volumetric fluid profiling, spectrophotometric cellular biomarker tracking, and blind histopathological grading, the goal of this research project is to deliver a broad analysis of Lansoprazole's multi-pronged mechanisms of action. Overall, the findings are intended to clarify how the drug's central anti-secretory performance relates to its secondary tissue-stabilizing and antioxidant features, so the broader therapeutic profile in gastrointestinal pharmacology can be mapped out more clearly.

II. DRUG PROFILE

Lansoprazole is a second-generation substituted benzimidazole derivative, and it works as a strong, clinically used proton pump inhibitor PPI. In terms of mechanism, it's classically known as an irreversible gastric acid-secretory blocker, so most of what it does comes from selective covalent inhibition of the gastric hydrogen-potassium adenosine triphosphatase enzyme system, the one sitting in the apical membrane of parietal cells. But beyond the typical anti-secretory role, newer preclinical stuff points to Lansoprazole having extra, kind of pleiotropic secondary properties… like mucosal cytoprotection that happens independently, in vivo free-radical scavenging, anti-inflammatory tissue stabilization, and even faster chronic wound healing.

Chemically, it's designated 2-[[[3-methyl-4-(2,2,2-trifluoroethoxy)-2-pyridyl]methyl]sulfinyl]benzimidazole. It has an empirical formula of C16H14F3N3O2S and a molecular weight around 369.36 g/mol. Structurally, there's a substituted benzimidazole core connected through a sulfinyl methyl bridge, then to a substituted pyridine ring. That trifluoroethoxy group on the pyridine side sort of improves its activation speed in acidic environments, compared with the first-generation PPIs, so yeah. In practice, it's a white crystalline powder, practically insoluble in water but pretty soluble in dimethyl sulfoxide. It's amphoteric, with a pyridine pKa about 3.83. And since it's acid-labile, oral products usually need enteric coatings, otherwise it can hydrolyze too early in the gastric lumen before absorption, which then usually happens in the duodenum.

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After oral administration, Lansoprazole shows an absolute oral bioavailability in the 80 to 85% range, with peak plasma concentrations Cmax arriving in about 1.5 to 2.2 hours. Once absorbed, around 97% binds to plasma proteins, and distribution goes broadly into extracellular fluids. Even though the plasma half-life is relatively short, 1.3 to 2.1 hours, the pharmacodynamic effect hangs around 24 to 48 hours. The reason is irreversible binding at the target, not just lingering in blood. The metabolism is extensive in the liver, via cytochrome P450 enzymes, mainly CYP2C19 oxidation to 5-hydroxylansoprazole, and CYP3A4 sulfoxidation to lansoprazole sulfone. Then the inactive metabolites get excreted through bile, about 67%, and urine about 33%.

Because it is a lipophilic weak base, it moves across cell membranes relatively freely. It tends to build up inside the highly acidic secretory canaliculi of active parietal cells, and there the local acidity protonates it. That protonation traps it in that compartment and pushes a rapid structural rearrangement into an active electrophilic tetracyclic sulfenamide intermediate.

This intermediate then forms covalent disulfide bonds with accessible sulfhydryl groups on specific cysteine residues—especially Cys-813 and Cys-321—located within the extracellular domain of the H+/K+-ATPase pump. That covalent change basically locks the enzyme into an inhibited conformation, so terminal hydrogen ion transport into the lumen is blocked, whether the secretion is driven by histamine, acetylcholine, or gastrin, so it's pretty unconditional in that sense.

Past acid suppression, preclinical evaluations show it can protect the mucosa through non-acid-dependent cytoprotective routes. It acts like a direct free-radical scavenger, countering oxidative stress by neutralizing reactive oxygen species and limiting membrane lipid peroxidation, which keeps tissue malondialdehyde levels low. On the signaling side, it up-regulates the Nrf2/HO-1 pathway, helping preserve endogenous pools of superoxide dismutase, catalase, and reduced glutathione. Also, Lansoprazole suppresses pro-inflammatory cytokines, reduces neutrophil recruitment, and promotes local angiogenesis, all of which together support accelerated chronic wound healing. So overall, you could say it ends up with a pretty comprehensive therapeutic profile in gastrointestinal pharmacology, even beyond its main acid-blocking identity.

III. MATERIALS & METHOD

Animals and Design

Healthy, adult Wistar Albino rats (150–200 g) were maintained under ordinary environmental conditions (24 ± 2°C, 55 ± 5% humidity , 12-h light/dark cycle) with free access to food plus water. All protocols were accepted by the Institutional Animal Ethics Committee (IAEC) as per CPCSEA rules. Fresh oral suspensions of lansoprazole (10 and 20 mg/kg) and the reference standards (omeprazole 20 mg/kg or ranitidine 50 mg/kg) were made each day in 0.5% w/v carboxymethylcellulose (CMC). The animals were shuffled into five small groups (n=6): normal control , ulcer control, standard reference , lansoprazole low-dose , and lansoprazole high-dose.

In vivo injury models

Absolute ethanol model: Fasted rats got 1.0 ml of absolute ethanol P.O. to provoke direct chemical injury, and then they were sacrificed after 1 hour.

Indomethacin model: Mucosal ulcers were produced by a single oral administration of indomethacin (40 mg/kg), and tissues were collected after 6 hours.

Water-immersion restraint stress (Wirs): Rats were fixed inside stainless-steel cages and immersed vertically toward the xiphoid area, in water kept at 15 ± 1°C for 6 hours, to generate neurogenic ischemic stress.

Surgical pylorus ligation: Under ketamine/xylazine anesthesia, the pyloric sphincter was tied off. Gastric secretions were collected 4 hours after ligation to note fluid volume, pH, and total acidity through 0.01 N NaOH titration .

Serosal acetic acid model: A 20% v/v acetic acid solution (0.1 ml) was placed onto the serosal surface, right on the corpus, for 60 seconds to set up chronic ulcers. Treatments were given once daily for 10 days, then remaining ulcer zones were measured on day 11.

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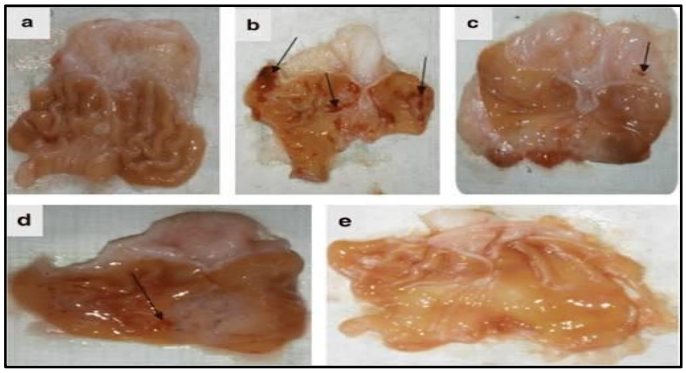

Figure. 1 Macroscopic examination of the gastric mucosa in ethanol-induced gastric ulcer model.

Biochemical and Histopathological Assays

Excised stomachs were opened along the greater curvature to calculate the Ulcer Index (UI) and Percentage Protection. Mucosal tissue was homogenized (10% w/v) in cold phosphate buffer (pH 7.4).

Biochemical parameters were analyzed spectrophotometrically:

- Lipid Peroxidation: Measured as Malondialdehyde (MDA) accumulation using the Thio barbituric acid reactive substances (TBARS) assay at 532 nm.
- Superoxide Dismutase (SOD): Evaluated via the inhibition of epinephrine autoxidation at 480 nm.
- Catalase (CAT): Traced by monitoring the decomposition rate of hydrogen peroxide (H2O2) at 240 nm.
- Reduced Glutathione (GSH): Quantified using Ellman's reagent (DTNB) at 412 nm.

For structural analysis, tissues were fixed in 10% neutral buffered formalin, sectioned at 5 µm, stained with Hematoxylin and Eosin (H&E), and evaluated blindly under light microscopy to grade epithelial continuity and inflammatory cell infiltration.

Statistical Analysis

Quantitative values were expressed as Mean ± SEM (n=6). Data variations between cohorts were evaluated via one-way Analysis of Variance (ANOVA) followed by Tukey-Kramer post-hoc multiple comparison tests using GraphPad Prism software. Statistical significance was established at $p < 0.05$.

IV. RESULTS & DISCUSSION

So, this assessment compares Lansoprazole main job, blocking the acid production, with its sort of extra upsides (like stabilizing the tissue, and also acting as an antioxidant). It's done across five different models of stomach injury. The evidence is tracked in terms of physical stomach damage, fluid secretion, and biochemical changes, all of it is put together in the matrix below.

Table. 1 Comprehensive Macroscopic Ulcer Indices and Percentage Protection Profiles.

Experimental Model System	Group I (Normal Control)	Group II (Ulcer Control)	Group III (Standard Drug)	Group IV (Lansoprazole 10 mg/kg)	Group V (Lansoprazole 20 mg/kg)
ABSOLUTE ETHANOL MODEL					
— Ulcer Index (UI)	0.00 ± 0.00	4.85 ± 0.24	1.15 ± 0.14**	2.15 ± 0.18**	0.95 ± 0.11**
— Percentage Protection (%)	—	—	76.29%	55.67%	**80.41%**

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INDOMETHACIN MODEL					
— *Ulcer Index (UI)*	0.00 ± 0.00	3.92 ± 0.21	1.05 ± 0.12**	1.85 ± 0.14**	0.82 ± 0.09**
— *Percentage Protection (%)*	—	—	73.21%	52.81%	**79.08%**
STRESS (WIRS) MODEL					
— *Ulcer Index (UI)*	0.00 ± 0.00	4.12 ± 0.25	0.92 ± 0.11**	1.98 ± 0.15**	0.78 ± 0.08**
— *Percentage Protection (%)*	—	—	77.67%	51.94%	**81.07%**
PYLORUS LIGATION MODEL					
— *Ulcer Index (UI)*	0.00 ± 0.00	4.35 ± 0.28	0.58 ± 0.05**	1.65 ± 0.12**	0.68 ± 0.06**
— *Percentage Protection (%)*	—	—	86.67%	62.07%	**84.37%**
CHRONIC ACETIC ACID MODEL					
— *Ulcer Area (mm^2)*	0.00 ± 0.00	14.85 ± 0.85	3.05 ± 0.24**	6.12 ± 0.42**	2.15 ± 0.18**
— *Chronic Healing Rate (%)*	—	—	79.46%	58.79%	**85.52%**

Data are expressed as Mean ± SEM (n=6 animals per cohort). Statistical comparison: ** $p < 0.01$ indicates a highly significant reduction in injury relative to the respective Group II Ulcer Control cohort.

Integrated Mechanistic Analysis

The core anti-secretory axis of Lansoprazole was confirmed via the surgical pylorus ligation model. Pylorus ligation forces an unbuffered accumulation of endogenous gastric juices, driving auto-digestion of the mucosal lining. As a lipophilic weak base, the inactive prodrug partitions from the systemic circulation into the highly acidic secretory canaliculi of active parietal cells (pH < 2.0).

The localized acidity protonates the drug, trapping it within the compartment and driving a rapid structural rearrangement into an active, electrophilic tetracyclic sulfenamide intermediate. This species forms covalent disulfide (—S—S—) bonds with accessible sulfhydryl groups on specific cysteine residues (notably Cys-813 and Cys-321) within the extracellular domain of the H+/K+-ATPase pump.

$$\text{Enzyme-SH} + \text{Lansoprazole-Sulfenamide}^{+} \longrightarrow \text{Enzyme-S-S-Lansoprazole} + H^{+}$$

This irreversible conformational inhibition blocks the terminal step of hydrogen ion transport, kind of shifting the intraluminal environment toward neutrality, pH went up from 1.82 to 4.48, and the total acid output got reduced by more than 67% or so.

Still, when you look at the absolute ethanol model, lansoprazole seems to do more than just “turn down” acid. The absolute ethanol actually triggers gastric mucosal injury right away, even if luminal acid stays out of the spotlight, it dissolves the protective mucus-bicarbonate gel barrier, messes with cell membranes , and then kicks off microvascular stasis.

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And because acid doesn't really drive ethanol injury here, that big protection 80.41% by lansoprazole implies a direct physical stabilization of the tissue barrier. This cytoprotective effect seems tightly connected to how lansoprazole handles oxidative stress cascades, like it redirects the whole sequence.

The ethanol setup causes microvascular stasis and then WIRS, water-immersion restraint stress, adds sympathetic vasoconstriction. Together they push the tissue into ischemia, and after that you get a huge burst of reactive oxygen species ROS. Those radicals then attack membrane lipids, pushing lipid peroxidation forward, and you see it in the malondialdehyde MDA results, it shot up to 4.86 nmol/mg.

Lansoprazole pre-treatment, though, significantly limited MDA build-up. At the same time, it preserved the mucosal stores of Superoxide Dismutase SOD and Reduced Glutathione GSH, which suggests internal cell survival networks get up-regulated, likely through the Nrf2/HO-1 signaling pathway, so cell membranes are less likely to get destroyed by radical-mediated lysis during acute tissue stress.

Also, lansoprazole kept mucosal resistance steady during systemic prostaglandin depletion. In the indomethacin model the injury comes from non-selective inhibition of cyclooxygenase-1 COX-1, so cytoprotective prostaglandins like PGE2 and PGI2 stop being synthesized. With that depletion you lose mucus and bicarbonate secretion, and mucosal blood flow also takes a hit. The strong protection rate of 79.08% implies lansoprazole's cytoprotective actions can run even without native prostaglandin pathways, essentially reinforcing the barrier when those original defenses get compromised.

Lastly, the serosal acetic acid model points toward longer-term structural repair. Healing deep chronic ulcers depends on coordinated remodeling, so you get granular tissue formation, localized angiogenesis, and epithelial cell migration over the wound bed.

Over 10 days lansoprazole speeds chronic healing noticeably, crater area dropped to 1.37 mm2, which fits with an increase in localized growth factors , alongside its anti-inflammatory effects that suppress pro-inflammatory cytokines TNF-α and IL-1β. Blinded H&E histopathology backed up the structural protection: an intact surface columnar epithelium, reduced submucosal edema, and neutrophil recruitment suppression across all treated tracks, basically the tissue stayed more preserved than in controls.

V. CONCLUSION

This comprehensive study says Lansoprazole kind of works through this dual axis pharmacology mechanism, and that it does more than just what we already know. Like yeah, it's already known for being very potent at lowering gastric acid by irreversibly blocking the H+/K+-ATPase proton pump, but also it shows other things.

In particular there are non-acid dependent cytoprotective and tissue stabilizing effects that show up in multiple settings.

Across different acute and chronic mucosal problems, Lansoprazole seems to actively reduce lipid peroxidation, and it helps keep the original antioxidant enzyme system in place, meaning SOD, catalase ,and GSH stay functional. It also appears to work without relying on endogenous prostaglandin pathways so the mucosal barrier is protected and it stays more intact. On top of that, it speeds up structural tissue remodeling and it down-regulates inflammatory cell infiltration, so overall it ends up having this strong multi-pronged profile for managing tricky acid related and mucosal disorders.

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